THE TREATMENT OF THE PERITONEUM IN DIFFUSE PERITONITIS.[1]

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FROM the stand-point of therapy, the classification of the purulent forms of peritonitis by the specific nature of the bacterial invasion is not of so much importance as that based upon the progress and extent of peritoneal involvement. Therefore, it has been my custom to classify these cases in the following groups: 1. Cases with abscess, in which there is a localized collection of pus with limiting adhesions. 2. Cases with spreading peritonitis, in which there is no limitation of the process by adhesions or gravitation, but in which the limits are ascertainable. 3. Cases of general peritonitis, in which no parts of the peritoneum, possibly excepting the lesser sac, can be demonstrated as free from the invasion. The last two groups are comprised under the general term, diffuse peritonitis.

In my report of cases I have held rigidly to this classification, and I have included in my cases of spreading peritonitis cases which would ordinarily be considered to be cases of general peritonitis. I have also excluded a number of cases which recovered and in which the process was spreading, but was not extensive. In other words, the cases of spreading peritonitis here presented would, in so far as I can judge from my experience, all have become generalized in a few hours, and further all presented the symptoms of a generalized peritonitis before operation. I particularly emphasize this point since these cases are of especial interest in regard to certain questions of treatment. All the cases have been operated upon by myself and comprise all the cases of general and spreading peritonitis arising from all causes that I have met with in the last two and one-half years, excepting those occurring in connection with new growths and intestinal obstruction where the primary disease overshadowed the secondary peritonitis.

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In three actually moribund cases—one from appendicitis and two from the perforation of typhoid ulcers—I have refused to operate, and these cases died within a few hours of the time I first saw them; but, on the other hand, I have been led to operate upon a number of moribund cases by the recovery of some cases that seemed positively hopeless.

It is not my aim to advance anything new in the treatment of these cases or to claim anything unusual in the way of results, but to simply report the cases and give the reasons that have led me to the treatment I now employ. I have chosen the cases occurring in my practice during the last two years and a half, since they have been operated upon under practically the same conditions, and therefore are better for comparison.

The cases have been tabulated and are appended. They comprise seventeen cases of spreading and fifteen cases of general peritonitis. Of the cases of spreading peritonitis, ten were caused by appendicitis, of which ten recovered and none died; one by perforating typhoid ulcer, no recovery, one death; four by perforating ulcer of the stomach or duodenum, of which two recovered and two died, a mortality of 50 per cent. The deaths were both attributable to sepsis from the local condition; two from gonorrhœal infection, both recovering from the peritonitis, but one dying later on the thirty-fifth day after operation from an obscure pyæmia. Of the cases of general peritonitis, seven were caused by appendicitis, of which three recovered and four died, a mortality of 57 per cent.; one by perforating typhoid ulcers, one recovery, no death; one by perforating tubercular ulcer, no recovery, one death; six by infection through the Fallopian tubes, one of which was probably a pneumococcus infection, one a mixed streptococcus and gonococcus infection, and the remainder gonorrhœal, of which one recovered and five died, a mortality of 83 per cent.

The latter six cases with the two cases of spreading gonorrhœal peritonitis form a rather remarkable series of eight cases of tubal infection and deserve a more extended description than I can afford them in this paper. I regret exceedingly that an absolute bacteriological diagnosis was only made in four of them; in three of the remainder I was led to believe that the infection was gonorrhœal from the clinical evidence. The high mortality was due largely to a condition of extreme sepsis; in one case death occurred from a postoperative volvulus apparently induced by a congenital anomaly of the large intestine.

In the treatment of these cases I have endeavored to carry out two principles: firstly, the removal or segregation as quickly as possible of the cause or nidus in order to prevent further peritoneal and systemic infection; secondly, the placing of the peritoneum under the best possible conditions to withstand and eliminate the generalized infection.

In regard to the propriety of the first principle, which in other words is early operation, I think there is no question, possibly excepting the cases of peritonitis arising from appendicitis.

I myself cannot see the difference between a case of extensively spreading peritonitis caused by infection from the appendix and one arising from any other cause, and further, I cannot believe that such an extensive peritonitis can be localized or arrested by any form of treatment other than operation.

I operate on these cases as soon as they can be prepared, believing that with the cause and the major portion of the toxic effusion removed the peritoneum is placed in a much better position to eliminate and destroy the remaining infection.

I think that any of the advocates of the treatment by rest will concede that the patient would be much better off with the appendix out, if that could be done without operation.

We have then simply to weigh the dangers of the additional toxæmia of the anæsthetic and the shock of operation against the danger of continued infection, both systemic and peritoneal, from the appendix itself or the escape of its contents.

With a short anæsthesia and a quick operation the danger of interference is much less than that of procrastination. There need be no fear of spreading infection, since that has been already accomplished. We have not so much the peritoneum to fear as we have the systemic infection through the lymphatics and vessels of the meso-appendix. Furthermore, I believe that an inflamed peritoneum is far better able to withstand an augmentation of the infection than a healthy peritoneum the scattered contents of a quiescent abscess. The proper time for the rest treatment in these cases is after the appendix is out and the peritoneum cleansed. I think the cases of spreading peritonitis caused by appendicitis which I report support my contention. These were all cases of extensive spreading peritonitis, ten in number, and all recovered. If we add the cases of general peritonitis in which the whole of the greater sac was demonstrated to be involved, seven in number, the total is seventeen cases operated upon with four deaths, a mortality of 23.5 per cent. Adding one moribund case dying without operation, the total mortality is 27.7 per cent.

The second principle involved in the treatment, namely, the placing of the peritoneum under the best possible conditions to withstand and eliminate the generalized infection, involves two main questions, namely, the cleansing of the peritoneum and the employment of drainage.

In regard to the cleansing of the peritoneum, I have consistently done it, employing for this purpose large quantities of decinormal saline solution at a temperature of 110° F., and consequently have nothing to say about the results obtained by not cleansing or by cleansing by other methods.

I have introduced the solution with a very large glass tube or have poured it in with a pitcher. In most instances I have partially dried the peritoneum afterwards with sponges or large rolls of gauze, or, on the other hand, have left considerable quantities of the solution in the cavity without apparent difference in the results. I am inclined to the latter procedure, since unnecessary handling of the intestines and prolongation of the operation are thereby avoided. My reasons for preferring washing are: that it can be carried out through a relatively small incision; that the peritoneal endothelium is less injured, and that the toxins are more readily diluted and removed. All loose fragments of fibrin are sluiced out, but the adherent portions are left.

In considering the questions of drainage the cases are best divided into groups, one including those in which the focal cause or origin can be removed or eliminated, the other the remaining cases.

To the first group would be assigned most peritonitis arising from perforation of the hollow viscera, from appendicitis, salpingitis, and possibly cholecystitis; to the second those resulting from rupture of abscesses, from pancreatitis, and cholecystitis in which cholecystectomy was contraindicated.

In the first group drainage may or may not be employed; in the second it is, as a rule, necessary.

I was formerly a warm advocate of abundant drainage; later I became convinced of the utter impossibility of draining every part of the peritoneal cavity, for it was evident that the drains were soon isolated by adhesions; so I next confined myself to the drainage of the field of operation, and then perceiving that the other similarly affected regions of the peritoneum took care of themselves, I have lately omitted drainage almost entirely, and only employed it when the presence of necrotic tissues or hæmorrhage has demanded it.

A comparison between the cases in which drainage was employed and those in which it was omitted is of interest in regard to the occurrence of complications and the duration of convalescence.

One of the principal advantages that non-drainage has over drainage is the avoidance of intestinal obstruction. Intestinal obstruction may be caused by the direct pressure of the drain on the intestine or by the production of adhesions about the site of the drain. Adhesions no doubt occur when no drains are used, but they are undoubtedly increased by their presence.

The only case in this series in which obstruction occurred which could be directly attributed to the drainage was Case 3, of spreading peritonitis following appendicitis. In this case a glass tube was used, passing to the pelvis, and the incessant vomiting and distention from which the patient suffered disappeared immediately on removal of the tube. Although this is the only accident of this kind appearing in this series, I have observed it several times in other cases. It has, however, seemed to me that there has been much less postoperative meteorism and vomiting in the cases not drained than has been the usual experience when drainage has been employed.

A not infrequently observed complication has been the formation of localized collections of pus in various parts of the peritoneum during convalescence.

This is apparently due to the inability of the vital forces to overcome the entire infection, and consequently more or less isolated patches of unresolved inflammation are left behind.

These collections do not always occur in the natural pockets or fossæ of the cavity, and so cannot be entirely explained by gravitation of the exudate. For the same reason they cannot be prevented by drainage as usually employed. These abscesses were observed in four cases, twice in drained and twice in undrained cases. In one drained case (13), a case of spreading peritonitis due to gastric ulcer, a subphrenic abscess developed from direct extension from the area of drainage in front of the stomach. Probably the aspirating movements of the diaphragm were much to blame for this. In the other drained case, a case of general peritonitis due to appendicitis, a pelvic abscess was opened on the thirty-seventh day after operation. In this case it might be attributed to the irritation of the drain, which passed to the pelvis, and its too early removal.

Both of the undrained cases developing late abscesses were cases of appendicitis; one with spreading peritonitis developed a subphrenic abscess; the other, an extreme case of general peritonitis, developed two abscesses in the fourth and fifth weeks of convalescence, situated among the coils of the intestines to the left of the umbilicus.

Drainage would probably not have prevented these abscesses. Of the ten cases closed without drainage four developed suppuration of the parietal wound. In one of these the superficial wound had been drained, but insufficiently. One other case of general peritonitis from the perforation of three typhoid ulcers of three days' standing, in which I had inserted a small cigarette drain into the pelvis, leaving it only for two days, also developed suppuration of the whole parietal wound, while the peritonitis went on to complete resolution.

When I close the peritoneum in these cases I now fully drain the parietal wound down to the peritoneum.

A comparison of the duration of convalescence in the drained with that in the undrained cases shows that the average duration of convalescence in all the drained cases was 43.4 days, in all the undrained cases 25.3 days,—an average saving to each of the undrained cases of eighteen days. One of the drained cases (25) is omitted, since his typhoid delayed his convalescence. A fairer comparison is obtained by taking only the cases of appendicitis with spreading peritonitis, since it so happens that all of these cases could have been treated without drainage and that the other conditions were practically alike. In this group the average duration of convalescence of the drained cases was 34.7 days, of the undrained cases 21.8 days, —an average saving to the latter of practically thirteen days.

In the same group of cases the average length of time elapsing before the temperature struck the normal was in the drained cases 8.5 days, in the undrained cases 3.8 days,—a very marked difference in favor of the latter, showing how much shorter septic absorption lasts in the cases treated without drainage.

A comparison of the mortality in the cases treated by drainage and those without drainage would obviously be unfair unless we group the cases according to the causative diseases, and even then is unsatisfactory because the numbers are so small.

The only considerable groups are the cases of spreading peritonitis arising from appendicitis, ten in number, all of which recovered, showing no difference; the cases of general peritonitis arising from appendicitis, seven in number, of which five were drained with four deaths, and two were not drained, both recovering; and the cases of general peritonitis of tubal origin six in number, of which five were drained with no recoveries, and one was not drained and recovered.

Two of the latter group were children, and, not wishing to remove the tubes, I drained the pelvis, hoping that the process would become localized, thus making fatal mistakes. I also erred in some of these cases in my diagnosis and made lateral instead of median incisions, thus hampering myself and prolonging the operation. I now make it the rule, when operating on females, to make, in all cases in which there is the least doubt in diagnosis, a median incision.

A *résumé* of the treatment that has given the best results in my experience may be briefly stated as:

1. Early operation.
2. Lavage of the peritoneum with large quantities of saline solution.
3. Closing of the peritoneal cavity without drainage unless the latter is absolutely indicated by the presence of nonabsorbable amounts of necrotic material.

TABLE OF CASES OF SPREADING AND GENERAL PERITONITIS.—SPREADING.

No.	Age and Sex.	Cause.	Duration.	General Condition.	Amount of Peritoneum inflamed.	Exudate.	Drain.	Postoperative Complications.	Temp. Normal.	Duration of Convalescence.	Result and Remarks.
1	27 M.	Gangrenous appendicitis.	72 hours.	Fair.	Lower half.	Seropus, fibrin.	Yes.	....	10th day.	30 days.	Recovered.
2	26 M.	Perf. appendicitis.	42 hours.	Bad.	Lower two-thirds.	Pus, seropus.	Yes.	....	10th day.	27 days.	Recovered.
3	29 M.	Gangrenous appendicitis.	48 hours.	Fair.	Lower half.	Seropus, fibrin.	Yes.	Intestinal obstruction from pressure of drain.	11th day.	43 days.	Recovered.
4	60 M.	Gangrenous appendicitis.	36 hours.	Fair.	Lower half.	Pus, seropus.	Yes.	....	3d day.	39 days.	Recovered.
5	21 M.	Gangrenous appendicitis.	30 hours.	Good.	Right half and pelvis.	Seropus.	No.	....	3d day.	16 days.	Recovered.
6	16 M.	Gangrenous appendicitis.	48 hours.	Fair.	Lower two-thirds.	Pus, seropus.	No.	....	3d day.	16 days.	Recovered.
7	23 F.	Gangrenous appendicitis.	24 hours.	Bad.	All, except about liver and spleen.	Pus, seropus, fibrin.	No.	....	4th day.	19 days.	Recovered.
8	11 F.	Gangrenous appendicitis.	24 hours.	Fair.	Right half and pelvis.	Seropus.	No.	....	3d day.	10 days.	Recovered.
9	25 M.	Gangrenous appendicitis.	36 hours.	Fair.	Lower two-thirds.	Seropus, fibrin.	No.	Superficial wound infection, subphrenic abscess.	4th day.	47 days.	Recovered.
10	23 M.	Gangrenous appendicitis.	26 hours.	Fair.	Lower half.	Seropus.	No.	....	6th day.	23 days.	Recovered.
11	36 M.	Perf. typhoid ulcer.	26 hours.	Bad.	Lower two-thirds.	Pus, fibrin, fæces.	Yes.	....	....	....	Died in two hours.
12	25 M.	Perf. ulcer of duodenum.	48 hours.	Fair.	All, except about spleen.	Pus, seropus, fibrin, intest. contents.	Yes.	....	15th day.	37 days.	Recovered. Inversion ulcer with purse string.
13	20 F.	Perf. ulcer of stomach, abscess; rupture of abscess.	20 hours.	Bad, anæmic.	Upper half.	Seropus, fibrin, gastric contents.	Yes.	Subphrenic abscess.	4th day.	....	Died tenth day, sepsis; peritonitis resolved.
14	21 F.	Perf. ulcer of stomach, abscess; rupture of abscess.	6 days, 12 hours.	Bad.	All, except small portion of lower regions.	Pus, seropus, fibrin, gastric contents.	Yes.	....	....	....	Died sixteenth day, sepsis from abscess.
15	47 M.	Perf. ulcer of pylorus.	7 hours.	Good.	Right side and pelvis.	Seropus, gastric contents.	No.	....	6th day.	22 days.	Recovered. Excision ulcer, pyloroplasty.
16	20 F.	Tubal, gonorrhœal.(?)	48 hours.	Fair.	All, except about liver.	Seropus.	Yes.	....	5th day.	33 days.	Recovered. No bacteriological examination.
17	57 F.	Tubal, gonorrhœal.	24 hours.	Good.	Right side of pelvis and right femoral hernial sac.	Seropus.	Yes.	....	2d day.	....	Died thirty-fifth day, pyæmia; temp. normal for three weeks after operation.

GENERAL.

No.	Age and Sex.	Cause.	Duration.	General Condition.	Amount of Peritoneum inflamed.	Exudate.	Drain.	Postoperative Complications.	Temp. Normal.	Duration of Convalescence.	Result and Remarks.
18	23 F.	Gangrenous appendicitis.	72 hours.	Bad.	Whole greater sac.	Seropus, fibrin.	Yes.	....	....	....	Died on third day.
19	18 M.	Gangrenous appendicitis.	72 hours.	Bad.	Whole greater sac.	Pus, fibrin.	Yes.	Pelvic abscess at seat of drainage 37th day.	28th day.	95 days.	Recovered.
20	38 M.	Gangrenous appendicitis.	....	Bad.	Whole greater sac; coils of intestine appeared gangrenous in places.	Pus, fibrin.	Yes.	....	....	....	Died on third day.
21	50 F.	Gangrenous appendicitis.	120 hours.	Bad.	Whole greater sac.	Pus, fibrin.	Yes.	....	....	....	Died. Only partial lavage; death in ten hours.
22	12 F.	Gangrenous appendicitis.	60 hours.	Bad.	Whole greater sac.	Pus, fibrin.	No.	Superficial wound infection, pneumonia 10th day, two abscesses left side 4th week.	40th day.	60 days.	Recovered. Streptococcus and colon bacillus.
23	25 F.	Gangrenous appendicitis.	48 hours.	Fair.	Whole greater sac.	Pus, seropus, fibrin.	No.	Superficial wound infection.	5th day.	20 days.	Recovered.
24	68 M.	Gangrenous appendicitis.	80 hours.	Moribund.	Whole greater sac.	Pus, fibrin.	Yes.	....	....	....	Died in three hours.
25	16 M.	Perf. three typhoid ulcers.	72 hours.	Bad.	Whole greater sac.	Pus, seropus, fibrin, fæces.	Yes.	Superficial wound infection.	3d day.	52 days.	Recovered. Small cigarette drain only; recrudescence typhoid fourth day.
26	24 M.	Perf. ulcer of ileum, probably tubercular.	48 hours.	Fair.	Whole greater sac.	Pus, seropus, fibrin.	Yes.	....	....	....	Died fourth day.
27	6 F.	Tubal, probably pneumococcus.	....	Bad.	Whole greater sac.	Creamy pus.	Yes.	....	....	....	Died ninth day; autopsy report pyæmia.
28	27 F.	Tubal, gonorrhœal.(?)	144 hours.	Bad.	Whole greater sac.	Pus.	Yes.	....	....	....	Died second day, sepsis.
29	30 F.	Tubal, gonorrhœal.(?)	36 hours.	Fair.	Whole greater sac.	Seropus, fibrin.	Yes.	Vulvulus 3d day.	....	....	Death followed second operation for intestinal obstruction; no bact. examination.
30	4 F.	Tubal, gonorrhœal.	....	Bad.	Whole greater sac.	Seropus.	Yes.	....	....	....	Died in twenty-four hours.
31	13 F.	Tubal, gonococcus and streptococcus.	72 hours.	Bad.	Whole greater sac.	Creamy pus.	Yes.	....	....	....	Died on fourth day from sepsis.
32	18 F.	Tubal, gonorrhœal.(?)	96 hours.	Fair.	Whole greater sac.	Pus, fibrin.	No.	....	....	20 days.	Recovered. No bact. examination.